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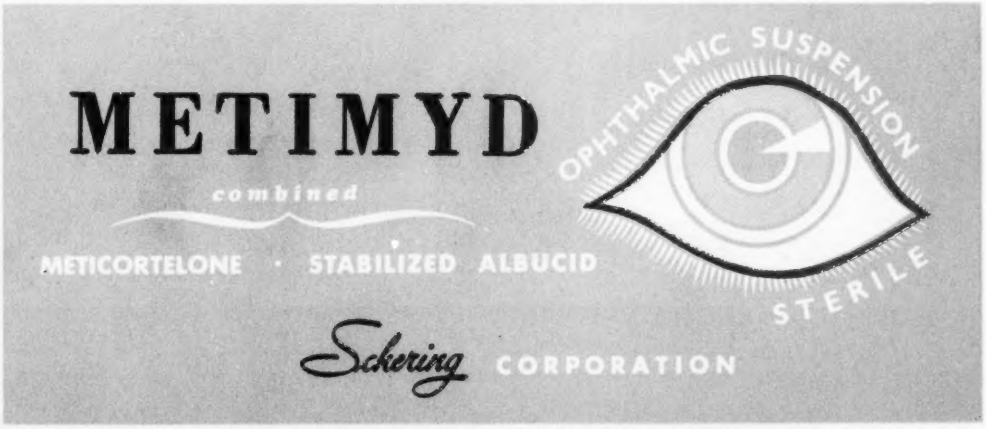

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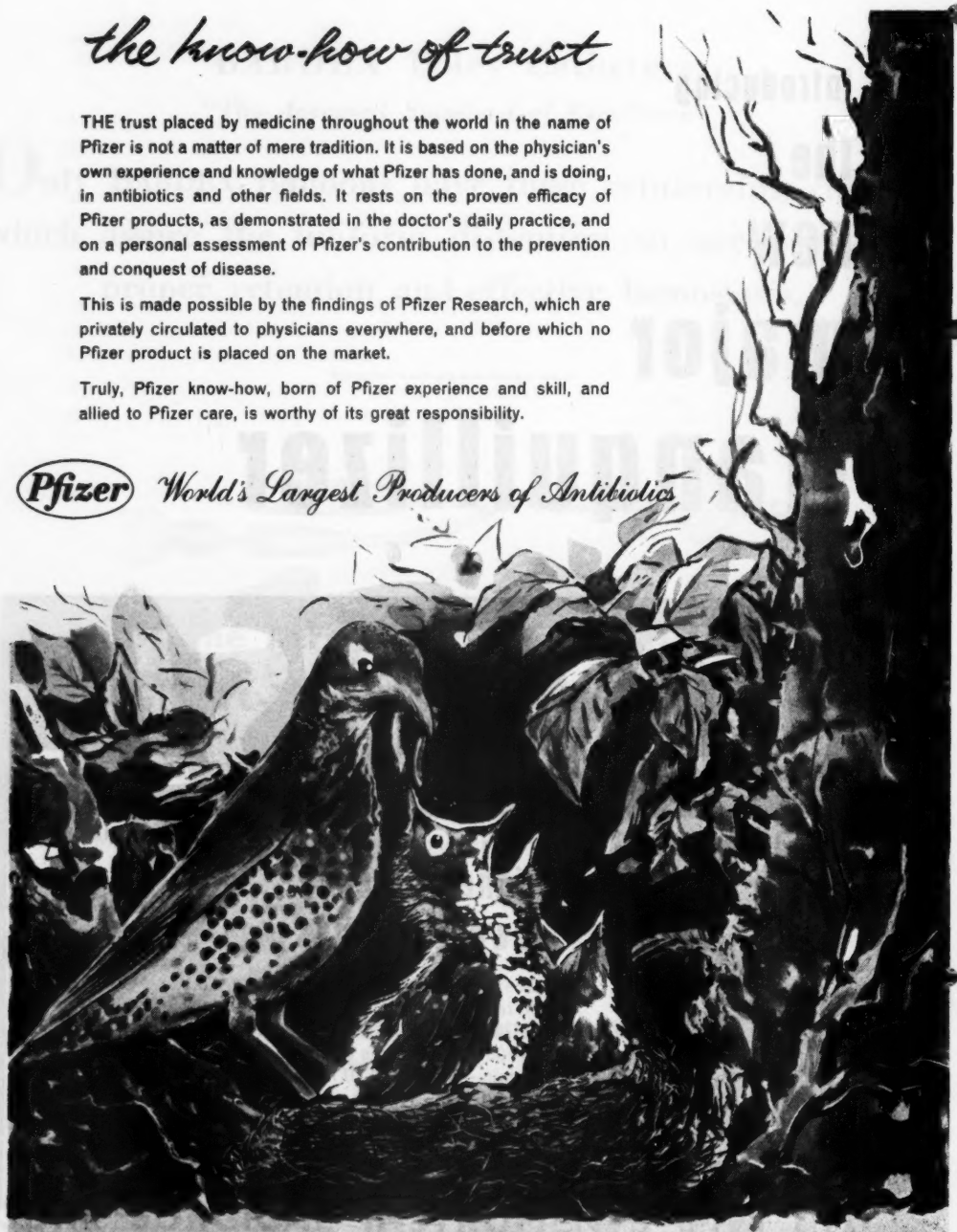

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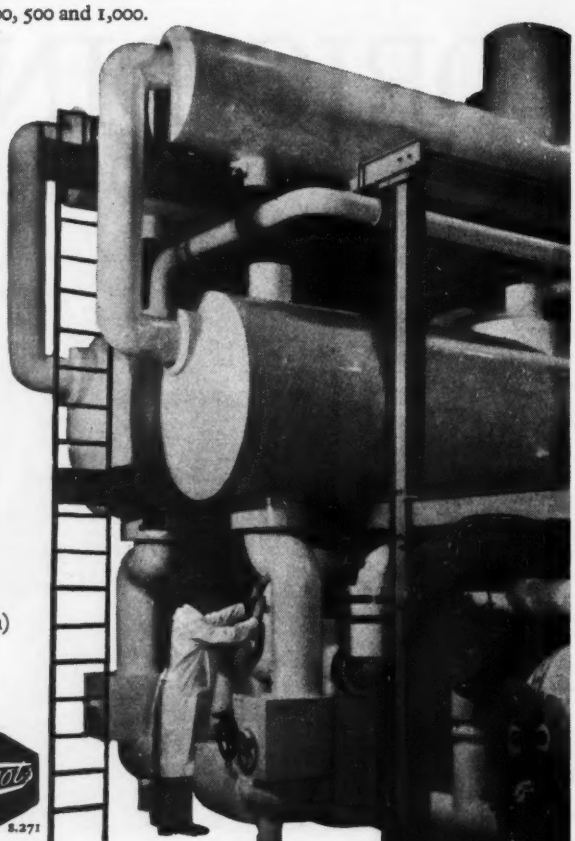

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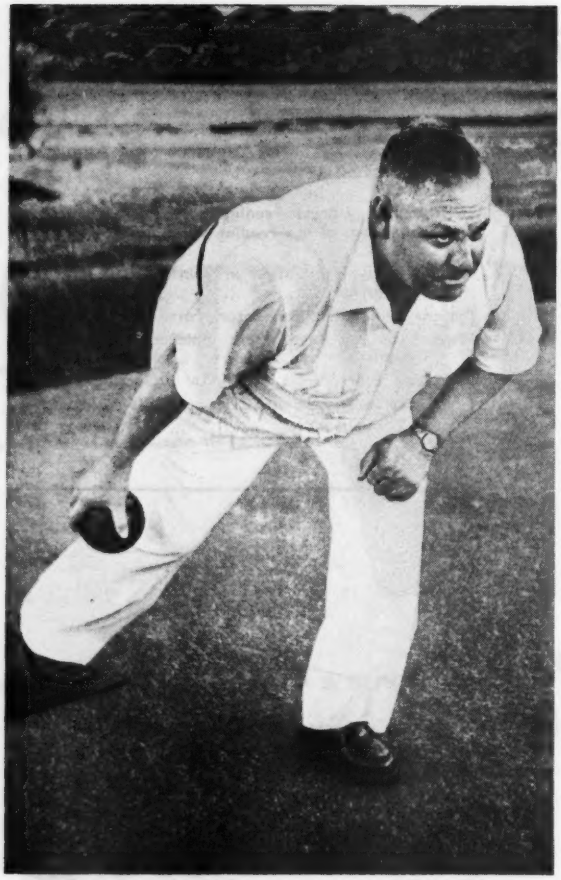

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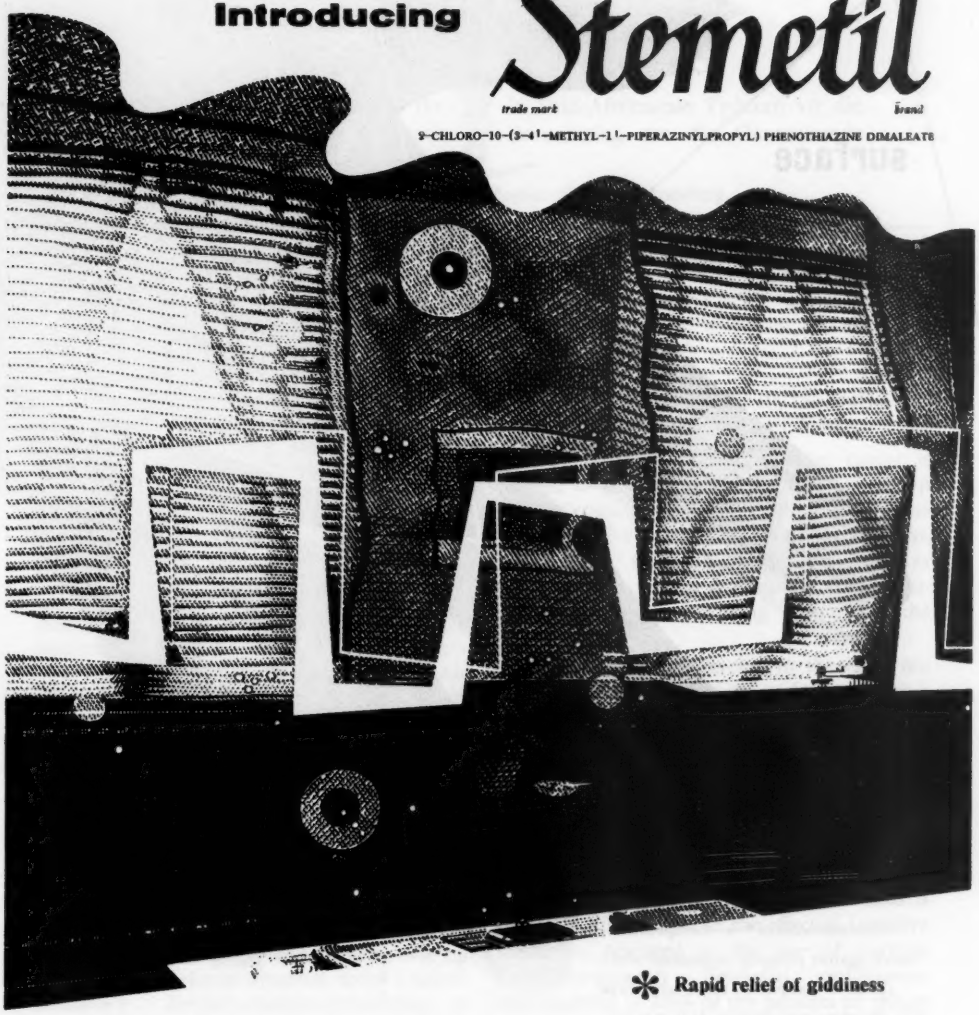

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REDAKSIONEEL · EDITORIAL

PULMONALE INFARKTE EN PULMONALE KARSINOOM

Daar word beweer dat die aansienlike toename in die aantal gevalle van longkanker in verband gebring kan word met die toenemende verbruik van tabak. Daar is egter ook ander oorsaaklike faktore wat in oorweging geneem behoort te word soos, byvoorbeeld, kwaadaardige degenerasie van littekenweefsel wat op dieselfde wyse as hepatosellulêre karsinoom voorkom volgende op sirrose van die lewer.

'n Karsinoom kan in die long ontwikkel nie alleen ten gevolge van tuberkulose-letsels nie, maar ook ten gevolge van die letsels wat deur infarkte veroorsaak is. Volgens die waarnemings van Baló *et al.*,* gebeur dit nog al dikwels, aangesien bloedsomloopkwale in die afgelope jare toegeneem het.

Aansienlike selvermenigvuldiging van die alveolêre epiteel kan op die grens van 'n hemorragiese infarkt plaasvind. Sodanige selvermenigvuldiging kan tegelykertyd in etlike foci ontstaan, en is die gevolg van anoksie of van die effek van prikkelende stowwe wat op die plek van nekrose vrygestel word (‚nekrohormone'). Epiteel-selvermenigvuldiging is waargeneem by 38% van die pasiënte wat aan lykskouing onderwerp is om pulmonale infarkte te bestudeer. Hierdie selvermenigvuldiging kan aanleiding tot 'n alveolêre selepiteel gee. In sekere omstandighede kan 'n plat selkarsinoom egter ook voorkom ten gevolge van metaplasie. Uitwendig ontwikkelde skadelike middels speel skynbaar geen rol in die degenerasie van littekenweefsel nie.

* Baló, J., Juhász, E. en Temes, J. (1956): *Pulmonary Infarcts and Pulmonary Carcinoma,* Cancer (V.S.A.), **9**, 918.

PULMONARY INFARCTS AND PULMONARY CARCINOMA

The considerable increase in lung cancer is alleged to be associated with a steep rise in tobacco consumption. There are, however, also other causative factors to be considered such as, for instance, malignant degeneration of scar tissue which occurs in a way similar to hepatocellular carcinoma following cirrhosis of the liver.

A carcinoma may develop in the lung, not only from tuberculosis scars, but also from scars of infarcts. According to the observations of Baló *et al.*,* this is quite often the case, especially as the incidence of circulatory disorders has increased in recent years.

Considerable proliferation of the alveolar epithelium can occur on the border of a haemorrhagic infarct. Such proliferation may arise in several foci at the same time and is the result of anoxia or of the effect of irritative substances liberated at the site of necrosis (' necrohormones '). Epithelial proliferation was observed in 38% of the patients on whom autopsies were performed for the study of pulmonary infarcts. This tissue proliferation may give rise to an alveolar cell epithelioma. In certain circumstances, however, a squamous cell carcinoma can also occur through metaplasia. Exogenous noxious agents do not, presumably, play any part in the degeneration of scar tissue.

* Baló, J., Juhász, E. and Temes, J. (1956): *Pulmonary Infarcts and Pulmonary Carcinoma,* Cancer, (U.S.A.) **9**, 918.

DIE ROOK VAN SIGARETTE EN KORONÊRE BLOEDVLOEIING

Die effek van sigaretrook op koronêre bloedvloeiing en hartspiermetabolisme is ondersoek deur middel van die regstreekse opmeting van volwasse vrywilligers met gesonde harte.* Een van die metodes waarvan gebruik gemaak is, was kateterisasie van die koronêre sinus sodat koronêre veneuse bloed verkry kon word.

Rook het 'n statisties betekenisvolle vermeerdering van die koronêre bloedvloeiing (van 70 tot 83 eenhede) tot gevolg gehad, terwyl koronêre bloedvaatweerstand aansienlik afgeneem het (van 130 tot 105 eenhede). Die polsslag het gemiddeld gestyg van 87 tot 93. Die hartspieruittreksel van suurstof en glukose wat aan koronêre bloedvloeiing verwant is, het in 'n min of meer opvallende mate verminder. Ten gevolge van die verhoogde bloedvloeiing deur die hartspier het die hartspierverbruik van suurstof en glukose per tyd-eenheid feitlik onveranderd gebly. Die uittreksel van laktate, piruvate en ketone was ook min of meer normaal.

Daar kan derhalwe aangeneem word dat die bloedvloeiing en die metabolisme van die gesonde hartspier nie deur die rook van sigarette belemmer word nie.

* Bargeron, L. M., jr., Ehmke, D., Gonlubol, F., Castellanos, A., Siegel, A. en Bing, R. J. (1957): Circulation, **15**, 251.

CIGARETTE SMOKING AND CORONARY BLOOD FLOW

The effect of cigarette smoking on the coronary blood flow and myocardial metabolism was investigated by direct measurement in adult volunteers with sound hearts.* One of the methods used was catheterization of the coronary sinus in order to obtain coronary venous blood.

Smoking produced a statistically significant increase in coronary blood flow (from 70 to 83 units), while coronary vascular resistance decreased considerably (from 130 to 105 units). The pulse rate rose on the average from 87 to 93. The myocardial extraction of oxygen and glucose, related to the coronary blood flow, declined to a more or less marked extent. As a result of the increased flow of blood through the heart muscle, however, the myocardial oxygen and glucose usage per unit of time remained practically unchanged. The extraction of lactates, pyruvates, and ketones was also more or less normal.

It may therefore be assumed that the blood flow and the metabolism of the healthy heart muscle are not impaired by cigarette smoking.

* Bargeron, L. M., jr., Ehmke, D., Gonlubol, F., Castellanos, A., Siegel, A. and Bing, R. J. (1957): Circulation, **15**, 251.

PATENT DUCTUS ARTERIOSUS

A REVIEW BASED ON SEVEN YEARS' EXPERIENCE OF 108 CASES

DAVID ADLER, M.B., CH.B., F.R.C.S., EDIN.
Johannesburg

On 24 October 1949 the first case of patent ductus, a child 5 years old, was operated upon in my Unit at the Transvaal Memorial Hospital for Children. I am indebted to Dr. Roseman of Pretoria for referring this child to me, to Dr. Bernard van Lingen, Head of the Cardiac Clinic of the Johannesburg General Hospital, for establishing the diagnosis, and to Dr. Paul Wood of London, who, whilst on a very busy lecture tour, kindly saw the child in consultation. It was they who strongly advised surgery and prised open some of the local medical portals for subsequent cardiac surgery. By June 1952 my own operated cases numbered 24 and these I reported in the *South African Medical Journal*.[1]

Since October 1949 we have seen 108 cases. Of these 9 have either refused or are awaiting surgery and 4 were operated on in the presence of a coarctation of the aorta, which was excised. The remaining 95 cases have been confirmed at operation and form the basis of this communication. Except for one Indian girl, all the cases were Europeans.*

HISTORICAL

Galen was aware of the patent ductus but the first anatomical description of this structure was by Carcano in 1574. The first clinical diagnosis of patency of the ductus was made as long ago as 1847 and was described at a meeting in London. Gibson in 1898 first described the pathognomonic machinery murmur that still bears his name. Operation was proposed by Munro in 1907, first attempted by Strieder[2] in Boston in March 1937 and successfully accomplished by Gross[3] of the same city in the following year. In Britain the first case was operated upon by Tubbs[4] on 5 December 1939. As far as I can ascertain, Prof. Charles F. M. Saint

* A further twelve cases have been operated upon since this paper was compiled.

performed the first operation upon a ductus in South Africa on 11 August 1943 on a little boy 7 years old.[5]

INCIDENCE

MacMahon[6] records an incidence of 3.2 congenital heart conditions in 1,000 live births; 1.6 were cases of patent ductus. It is stated that patent ductus occurs in about 1 : 5,000 or 1 : 6,000 persons. On this basis there must be some 500 cases of patent ductus amongst the European population of South Africa at present. This figure should be compared with 155 cases seen by Nicks[7] in 7 years in Auckland, New Zealand, drawn from a population of 2 million people.

The Cardiac Clinic of the Johannesburg General Hospital[8] has records of 110 cases of patent ductus seen between 1949 and December 1955; of these 79 are included in this series. In this 7-year period, of some 429 cases of congenital heart disease seen at the Cardiac Clinic, 336 were cases of acyanotic heart disease. The Cardiac Clinic at the Groote Schuur Hospital has records of 94 cases of patent ductus, of which 70 have been operated upon.[9]

From May 1948 to September 1956 76 cases of patent ductus were seen at the Thoracic Surgical Unit at Baragwanath.[10]

Aetiology. (*a*) *Heredity.* Although it was felt originally that heredity played no role in the causation of patent ductus, this has been shown to be incorrect. There is a greater tendency for a mother who has had one child with a congenital heart to have another child with a congenital anomaly and it is likely that this child will suffer from the same anomaly as its sibling.[11] A family history of patent ductus is commoner than with other congenital cardiac anomalies. I have operated upon 3 sibling girls (aged 6 years, 3 years and 17 months) for patent ductus.

(*b*) *Maternal rubella* is likely to result not only in patent ductus but in a wide short ductus. Gardiner,[12] from Australia, reports 3 broad ducts all from mothers with rubella; and Hultgren[13] states that 2 of his 8 atypical cases were similarly affected.

(*c*) *Sex Incidence.* Of a total of 95 cases of patent ductus, 31 (32.6%) were males and 64 (67.4%) were females (Fig. 1).

This is in striking contrast to coarctation of the aorta, aortic stenosis and pulmonary atresia, all of which are far commoner in males.

(*d*) *Age* (Fig. 2). As the patent ductus is a persistence of the normal foetal circulation, it is strange that it has been previously stated that it is rare to hear the typical murmur in infancy. This has not been so in a large number of our cases in which we have records of a typical machinery murmur heard in the first few months of life. Our youngest case was operated upon at the age of 4 months. The reports of operations on infants, however, appear to be relatively few, e.g. Cade's youngest case from Bristol was 2 years.[14] In Potts review of 241 cases,[15] the youngest was 10 months. Because in infancy the pulmonary arterial pressure is relatively high and the aortic pressure is relatively low, in many cases only a systolic murmur is heard. With age the aortic pressure rises and the pulmonary pressure falls. Flow then occurs from the aorta to the ductus both during systole and in diastole and the continuous murmur develops. Only 8 cases are reported in the literature over the age of 65 (the oldest, according to Ekstrom,[16] being 75). All had small ductuses.

It is of interest that before 1952 our youngest patient was 2¾ years, and this was the only case under 3 years of age. In the past 2 years we have operated upon 15 cases under the age of 3 years. This recent tendency to operate upon the young age group is world wide. In this younger age group the diagnosis is often difficult and the risk of delaying surgery so great if the ductus is large that this subject forms the basis of a further paper in the press on *The Patent Ductus in Infancy.*

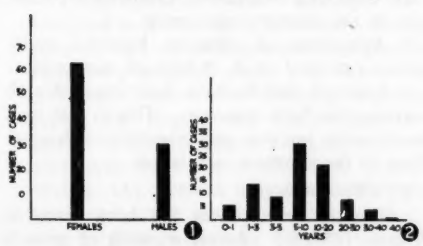

Fig. 1. Sex incidence. *Fig. 2.* Age incidence.

PROGNOSIS

The prognosis in untreated cases is difficult to assess but Keys and Shapiro[17] found that the life expectancy of patients with patent ductus was reduced by 25.5 years.

MacMahon[6] reviewed all cases of congenital heart disease born in Birmingham over a 10-year period. He found that of the 62 cases of patent ductus in infancy, 19 died of congestive cardiac failure or of its sequelae.

It seems likely, therefore, that patent ductus is the cause of considerable mortality in the

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first year of life and that there is then a period of relative freedom from symptoms for a large number of cases. With the passage of time, however, the vast majority will develop persistent symptoms. Shepherd,[18] from an analysis of 34 cases, reports that there is a slow, progressive increase of pulmonary arteriolar resistance with age. There is little justification, therefore, for a watching brief in cases of patent ductus arteriosus.

All authorities agree that at present the prognosis in untreated cases cannot be assessed with certainty. Very few cases, however, are found in middle-aged or elderly persons.

PATHOLOGY

The ductus is constant only in site, i.e. it passes from the pulmonary artery upwards to the aorta, joining the latter below the origin of the left subclavian artery. In cases with large shunts and gross pulmonary hypertension the ductus has differed in direction in that it has appeared to be a continuation of the left pulmonary artery. Its diameter and length vary greatly, being smaller and longer in infancy and becoming broader and shorter in adult life. This anatomical change should be kept in mind when surgery is elected, as it is obviously easier to operate in the younger age group than in adult life.

The pathological complications that can ensue are:

1. Physical retardation (extremely common in this series) from the deficient peripheral circulation resulting from the large shunt of blood from the systemic to the pulmonary circuit.
2. Left ventricular failure from the increased ventricular output. According to Keys and Shapiro,[17] very few survive once cardiac failure supervenes.
3. Right ventricular failure from the increased pulmonary hypertension.
4. Pulmonary arterio-sclerosis secondary to pulmonary hypertension.
5. Subacute bacterial endocarditis, which occurs mostly in the young age group.

Scheef[19] described a premature baby aged 11 days with umbilical infection and endarteritis. On the other hand Touroff[20] operated upon a woman of 63 with subacute bacterial endocarditis. Clagett[21] reports an incidence of 3.6%. Gelfman and Levine[22] stated in 1942 that 25% of patients with patent ductus die of infection, usually *Streptococcus viridans*, in association with dental extraction, infection of the upper respiratory tract and sometimes after parturition. We have had an incidence of 8.4%.

It is of interest to recall that the first recorded attempt at ductal surgery (on 16 March 1937) by Strieder[2] was on an infected case. The first such success and the first operation in England was that of Tubbs[4] on 5 December 1939.

6. Rupture of an aneurysm of the ductus.
7. Pressure on the left recurrent nerve.

DIAGNOSIS

Before 1952, and in keeping with the literature of that time, we found that 96% of our first series of cases had a typical Gibson murmur. In this further series of 72 cases only 59 (83%) had the typical murmur. The presence of the Gibson murmur over the pulmonary artery is pathognomonic of a patent ductus and calls only for corroborative evidence to make the diagnosis certain. However, a large number of big ductuses will not be diagnosed if those showing a systolic murmur only over the pulmonary artery associated with a high pulse pressure and signs of pulmonary plethora, are not adequately investigated.

The diagnosis then depends on the symptomatology, a careful physical examination and the elucidation of certain corroborative signs.

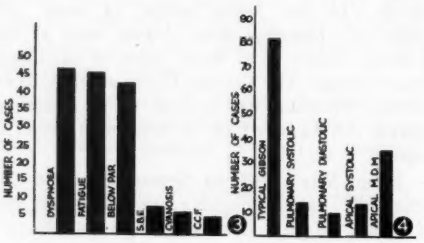

Fig. 3. Symptoms. *Fig. 4.* Murmurs.

(*a*) *Symptoms* (Fig. 3).

i. Dyspnoea was present in 47 (49.5%) of our 95 cases. Dyspoea varies and can date either from infancy or can develop later in life after a period of normal activity. Several of our adult cases have played rugby, only to develop breathlessness subsequently.

ii. Lassitude and fatigue are commonly experienced. Many parents have thought their children indolent, and only after surgery have they noticed a dramatic improvement in these symptoms. Forty-six (48.4%) of our cases presented with fatigue.

iii. Repeated respiratory infections are common in the younger age group.

iv. Symptoms of subacute bacterial endocarditis occurred in 8 (8.4%) of our cases.

v. Syncopal attacks have been rare, though dizziness has been common. This is due to a low diastolic pressure and relatively inadequate filling of the coronary circulation.

(*b*) *Physical Signs.*

i. Physical development has been tardy in 43 cases (45.3%). Actual stunting of growth has been frequent. In this type of case there

has been, without exception, dramatic gain in weight and in height in the first post-operative year.

ii. The colour is almost invariably good in the uncomplicated case. Pallor, however, occurred in those suffering from subacute bacterial endocarditis. Cyanosis due to a reversed shunt (i.e. when the pulmonary artery pressure exceeds the mean aortic pressure) was present in 7 cases (7.4%). These demonstrated differential cyanosis, i.e. the cyanosis of the toes was greater than that in the fingers. This phenomenon occurs as the result of a bi-directional, intermittent shunt of blood from the pulmonary artery to the aorta. Most of this de-oxygenated blood is directed into the aorta and into the lower extremities with an oxygen concentration less than that which goes to the upper limbs. The presence of a lower oxygen saturation in the femoral compared with that found in the brachial artery is diagnostic of a partial reversed shunt, and was present in 6 cases.

(*c*) The heart size is stated to be normal and this is so in the asymptomatic cases. Left ventricular thrust is, however, common when left ventricular hypertrophy takes place and many a mother has brought her child in for attention because, on holding the baby, she has felt the bounding character of the heart beat. In those cases of pulmonary hypertension characterized by right ventricular hypertrophy the classical left parasternal heave of the enlarged right ventricle is clinically palpable.

(*d*) The pulse pressure is high and is typically water-hammer in character. A palpable thrill is felt over the pulmonary artery in more than half the cases. In some, where the ductus is large, the thrill is felt all over the praecordium. Aitken[23] has drawn attention to the fact that in a broad, short, window-type of ductus the thrill is palpable in the suprasternal notch. This has been confirmed in our cases.

(*e*) *The Murmur* (Fig. 4).

i. The classical Gibson murmur may be described as a machinery or train-in-the-tunnel murmur and is pathognomonic when heard over the pulmonary artery. It is continuous throughout systole and diastole, accentuated during the former and diminishing in the latter. It is best heard over the pulmonary artery and below the middle third of the left clavicle. On occasion it is more widely distributed and rarely heard at a lower site.

In our first 24 cases the murmur was present in all but one (96%). In our subsequent 71 cases the murmur was absent in 12 cases, i.e. the classical murmur was present in only 83%. In this group of 71 cases 1 case in 6 had an atypical murmur. In the total of 95 cases 1 : 8 had an *atypical* murmur. As this is the type of case that has already developed pulmonary hypertension, it needs urgent investigation.

ii. In 14 cases where gross pulmonary hypertension has been proven, a harsh systolic murmur only was heard over the pulmonary artery.

iii. In 10 cases there was in addition an early diastolic murmur along the left parasternal border due to pulmonary incompetence. In several cases this has persisted after ligation.

iv. In 14 cases a harsh, apical systolic murmur was audible and in a few cases this has persisted after operation.

v. In 36 cases (38%) of the series, there was a mid-diastolic murmur at the apex. In the vast majority this disappeared after surgery. Some, however, took a little time.

McKusick[24] stated in 1956 that an apical mid-diastolic murmur is *occasionally* heard in patent ductus arteriosus due to relative mitral stenosis from torrential mitral flow and dilatation of the left ventricle. Nadas[25] states that the Lutembacher syndrome is a very rare cause of a mid-diastolic murmur and that it occurs frequently in all large left-to-right shunts. In several of our early cases there were doubts about a co-existent mitral stenosis.

Cournand[26] states that an apical mid-diastolic murmur is due to increased flow of blood from the lungs to the left side of the heart with dilatation of both chambers, and swirling of the blood through an organically normal valve into the enlarged left ventricle.

vi. Two of our cases had concomitant aortic stenosis. In 2 other cases an unsuspected ventricular septal defect became apparent after ligation.

(*f*) *The Electrocardiogram.* The ECG is stated to be essentially normal in patent ductus. This may be true of the typical asymptomatic case but is certainly not true of any large series. More cases of pulmonary hypertension with atypical murmurs are now seen. These, of course, have right ventricular hypertrophy due to the elevation of the pulmonary artery pressure and resistance. Where pulmonary hypertension is extreme, it equals or even exceeds the aortic pressure and right ventricular hypertrophy on the electrocardiogram is even more evident.

In our first series of 24 cases there was only one patient (4%) with ECG evidence of right

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ventricular preponderance. In our subsequent 71 cases there have been 8 cases (11.3%). It should be recalled that only a few years ago no less an authority than Gross[26] stated that in 525 cases he had only seen right preponderance in 6 cases and that in the presence of right ventricular preponderance on the ECG one should strongly suspect some other anomaly. Only 37 of our cases had normal ECG's, 28 showed left ventricular hypertrophy, 9 right ventricular preponderance, 12 had combined ventricular hypertrophy, several had right bundle branch block, left atrial preponderance and septal Q waves (Fig. 5).

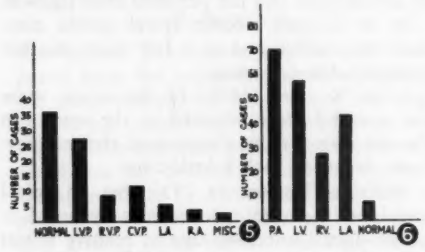

Fig. 5. E.C.G. findings. *Fig. 6.* Radiological findings.

(*g*) Although it used to be stated that most cases of patent ductus had no abnormal radiological findings, this has not been confirmed in my experience. Only 8 cases (8.4%) have been absolutely normal on careful radiological examination.

The abnormal radiological findings (Fig. 6) were:

i. Left ventricular hypertrophy (in 58 of our cases). This is an index of the increased left ventricular output.

ii. Enlargement of the pulmonary artery segment (in 71 cases). This varied from slight enlargement (Fig. 7) to immense enlargement of the pulmonary artery simulating aneurysm (Fig. 8).

iii. Enlargement of left atrium (Fig. 9) in 44 cases. This is due to increased pulmonary flow.

iv. Enlargement of the right ventricle in only 4 of the first 24 cases (16%) and in 24 of the next 71 cases (33.8%).

(*h*) *Cardiac Catheterization.* Although in most recently published series few cases were subjected to cardiac catheterization, I believe this is because few atypical cases are included. In our first 24 cases 8 were catheterized, mostly to confirm a clinical diagnosis of patent ductus. In Case No. 4 (published by Bothwell *et al.*[28]) there was gross pulmonary hypertension in the presence of only a pulmonary systolic murmur and there was differential cyanosis with the presence of a large shunt. Cardiac catheterization was performed in 23 of our subsequent 71 cases and in 17 of our last 24 cases. These include a fair number of large shunts in infants and babies under the age of 3 in whom there was only a systolic murmur. Catheterization is of value not only in confirming the presence of a patent ductus and excluding other pathology, but also in demonstrating very accurately the physiological changes in pulmonary artery pressure. The presence, then, of pulmonary hypertension, the direction of the shunt and whether it is bi-directional, transient or permanent, can be proved. All typical cases of patent ductus have a shunt from the aorta to the pulmonary artery with, in childhood, a relatively normal pulmonary pressure. In infants, however, pulmonary hypertension is more common and probably accounts for the higher percentage of congestive cardiac failures seen in this age group.

In a variable number of adults secondary pulmonary hypertension either develops or becomes evident. This group of cases becomes markedly incapacitated. As the pulmonary hypertension becomes extreme, the shunt becomes bi-directional, i.e. the pulmonary artery pressures are intermittently higher than those of the aorta. This occurs, e.g. in the presence of pain, on exercise and with anoxia. In these cases occasional cyanosis is present and in them cyanosis is more evident in the toes than in the fingers. (The explanation for this has already been discussed under *physical signs*). When the pulmonary artery pressure constantly exceeds that of the aorta, the patient is permanently cyanosed. We have had 22 cases of pulmonary hypertension with pulmonary systolic pressures above 45 mm. Hg.

The question whether a ductus ought to be operated on in the presence of pulmonary hypertension is controversial and forms the subject of a further communication in which we urge operation except in the reversed shunt.

(*i*) *Angiocardiography.* We have used this procedure only once (in Case No. 22). In infants[29] a retrograde aortogram through a catheter in the left radial artery or a Seldinger catheter passed down through the left common carotid is of value in demonstrating simultaneous filling of the aortic arch, the ductus and the left pulmonary artery. Cardiac catheterization, however, is of greater value as it not only proves the existence of a shunt but also its dimensions and its direction.

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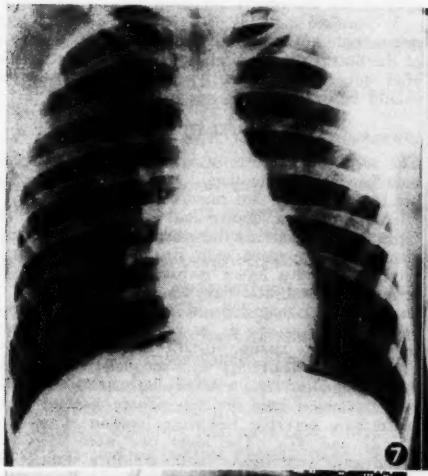

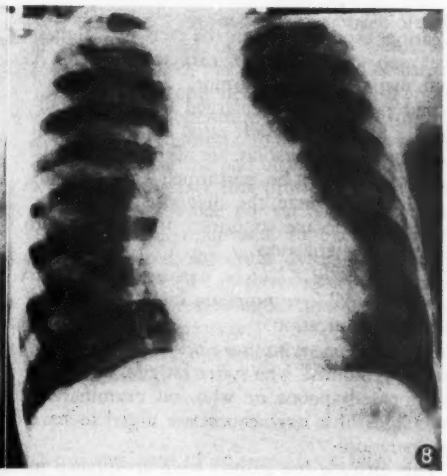

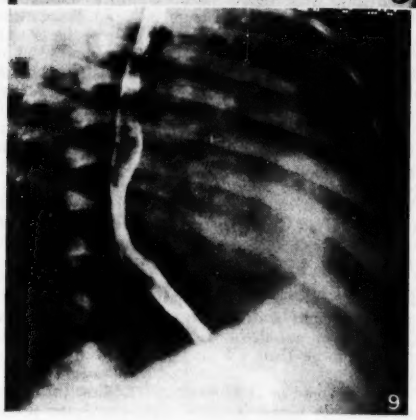

Fig. 7. X-ray demonstrating slight pulmonary artery enlargement.
Fig. 8. X-ray showing marked enlargement of the pulmonary artery.
Fig. 9. X-ray showing left atrial enlargement on the barium filled oesophagus.

(*j*) *Phonocardiography.* This is useful in recording the murmurs and for factual comparison before and after surgery. In complicated cases it is of considerable value.

DIFFERENTIAL DIAGNOSIS

The presence of a typical Gibson machinery murmur over the pulmonary artery is pathognomonic, but similar murmurs can be heard over arteriovenous aneurysms of the chest wall or of the lung. The Gibson murmur can be mistaken for a venous hum, but the latter disappears when the external jugular vein is compressed and its intensity varies with posture and with turning the head. The atypical cases must be differentiated from:

(1) Ventricular septal defect.
(2) Atrial septal defect.
(3) Rupture of an aneurysm of the sinus of Valsalva.
(4) Idiopathic pulmonary hypertension.
(5) An aortic pulmonary fistula. This can be most difficult. A very large shunt, a rather louder and more medial murmur than usual and gross pulmonary hypertension confirmed by catheterization during which the catheter passes into the aorta much more medially than in the usual ductus, confirm the diagnosis. We have had one such case with the pre-operative diagnosis of a ductus with pulmonary hypertension.

INDICATIONS FOR SURGERY

In 1952 the indications for surgery were strictly limited, as in most centres paediatricians and cardiologists were apprehensive and uncertain about recommending a prophylactic operation in childhood to prevent complications and morbidity in adult life. This conservative attitude has changed everywhere following the realization that:

1. Very few cases of patent ductus are asymptomatic.
2. The life expectancy in indubitably shortened, as very few cases are recorded over the age of 50.
3. Operation in the younger age group is comparatively safe with a mortality of about 1% in experienced hands.
4. Operative mortality is higher and the results are less satisfactory in the older age group in whom permanent right ventricular and pulmonary artery changes have already occurred.
5. Craffoord's[31] statement that he had 8 deaths in 16 cases of pulmonary hypertension and only 2 deaths in 266 typical cases suggested that the former group should not come to surgery. This is not our experience and I believe that the mortality can be reasonably low even in this group.

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1. *Surgery in Infants and Babies up to 3 Years Old.* In this age group the presence of sweating, especially about the face, is a suggestive sign of congestive cardiac failure. A pulmonary systolic murmur, an accentuated second pulmonic, an enlarged heart with pulmonary plethora and left atrial enlargement on radiological examination, are all danger signals. Operation should be performed early in this type of case where the diagnosis of a large ductus is made and confirmed, if necessary, on cardiac catheterization.

2. *Surgery in Children between 3 and 16 Years.* (*a*) All asymptomatic cases are advised to have an operation.

(*b*) Children who are below par, whose growth is stunted, who suffer fatigue, who have exertional dyspnoea or who, on examination, have signs of a large shunt, are urged to have an operation.

This attitude has gained wide acceptance in the past few years.

(*c*) All cases of pulmonary hypertension and atypical murmurs should be catheterized and, if there is gross pulmonary hypertension, surgery is imperative.

(*d*) Subacute bacterial endocarditis should be treated intensively with antibiotics for 3 weeks. Surgery must then be performed or recurrence of infection will take place.

3. *Surgery in Adults below 40 Years.* The foregoing criteria are still valid, but the operative mortality is likely to be higher and the relief of symptoms not quite as dramatic and satisfactory.

4. *Surgery in Adults over 40 Years.* In this group the risk of subacute bacterial endocarditis is less and, where no pulmonary hypertension has yet developed, I believe that surgery should be more carefully and individually assessed. Arteriosclerotic changes in the ductus wall itself and in the adjacent aorta and pulmonary artery, with calcification at the former site,[30] adds to these operative risks. Our oldest case was 43 years.

CONTRA-INDICATIONS TO SURGERY

1. When the patent ductus is a compensatory mechanism and is essential for the maintenance of adequate pulmonary circulation, e.g. patent ductus in the tetralogy of Fallot.

2. When the shunt is reversed, i.e. when the pulmonary arterial pressure constantly exceeds that of the aorta, the direction of the shunt will be from pulmonary artery to aorta. In this type of case right ventricular failure is likely to ensue. Most authorities therefore believe that the reversed ductus is a safety valve allowing some of the gross pulmonary hypertension to dissipate itself into the systemic circulation.

3. Standard methods of ligation or division ought not to be employed in the presence of an aneurysm of the ductus or when a false aneurysm has occurred after surgery. In this type of case hypothermia should be available.

OPERATION

The surgical treatment of a patent ductus will not here be described in detail, but it should suffice to say that there are two methods of treatment:

i. Division and suture has been popularized by Gross,[27] who believes that there is at least a 10% chance of recurrence with ligation. It should be recalled that this figure is based on his own early series when umbilical tape was used for ligation. I am convinced that division is the ideal procedure and that it is as safe as, or even safer than ligation in very expert hands. In the average clinic, however, ligation (carefully and adequately performed) is safer and carries a very low recurrence rate. Of our 95 cases 4 were divided. Three cases of recurrence have occurred following ligation (Case Nos. 25, 27 and 37). The latter two were short and broad and were ligated with umbilical tape. The third was a case with subacute bacterial endocarditis and pulmonary infarcts, and in which there were gross vegetations at both ends of the ductus. This ductus re-canalized within 10 days of surgery, probably because of peri-ductal oedema. I subsequently re-explored the patient and tied the ductus off completely. In 3 of our cases the ductus was so large that it was impracticable either to divide or ligate completely; but partial ligation resulted in 80–90% reduction in the shunt and in several of them a typical Gibson murmur was heard for the first time after surgery. Our recurrence rate is thus 3 in 92 cases (3.3% of the total). Many authorities agree that ligation is preferable, e.g. Crafoord,[31] Blalock,[32] Cade,[14] the Mayo Clinic[21] and others. There is no doubt that in a large ductus, ligation, as with division, is fraught with danger to such an extent that Gross[27] does not divide a patent ductus whose diameter is more than 1.5 cm., i.e. a circumference of 4.7 cm.

ii. *Ligation in Continuity.* I described this operation in detail in 1953, but there are several points worth reiterating and several new points in technique have emerged. It is essential to have adequate exposure. For this reason we prefer the postero-lateral incision with resection of a rib which gives excellent access to the ductus and to the aortic arch, with better control of any complications that may occur.

Until recently we have exposed the ductus by going anterior to the vagus nerve, but the aortic end can be more readily mobilized by dividing the mediastinal pleura posterior to this nerve. I believe that sharp dissection with scissors is preferable to blunt dissection and that there is less risk of damaging these arterial structures if one sees and cuts the peri-ductal adventitia under vision. Adequate length is obtained before ligation.

We have discarded umbilical tape as a suture material because sufficient tension cannot be obtained. Since a visit to Sir Russell Brock in London early in 1955, we have used Arfonad to lower the blood pressure during actual ligation. With systemic hypotension the vessels are lax and the ductus can be tied more readily. This was very evident recently in a child whose ductus was 2.5 cm. in circum-

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ference. I routinely use pleural drainage as in other thoracotomies, and have no reason to regret this precaution.

Four of our cases in this series were divided—one by myself and 3 by an assistant.

COMPLICATIONS OF SURGERY

1. *Haemorrhage at the time of operation.* Case No. 13 died from haemorrhage at the aortic end of the ductus during ligation.

2. *Damage to the left recurrent nerve.* This occurred in one ductus which was divided. Transient hoarseness occurred in Case No. 2.

3. *Ligation of other structures,* e.g. the left pulmonary artery. This seems impossible in skilled hands, but cases have been reported.[33, 34] In one of these cases (aged 11) the left pulmonary artery had been ligated in error six years before elsewhere. I have seen such a case locally. Meade[36] records a case where the artery was tied to control haemorrhage.

4. *Hyperpyrexia* is a risk in infants.

5. *Infection.* There has been one case of minor wound infection in an infant aged 17 months.

6. Two cases have required post-operative bronchoscopy for *left pneumonic atelectasis.*

7. *Right ventricular failure* occurs in cases of severe right ventricular hypertension and in cyanosed cases.

8. *Recanalization.* Three of our cases have recurred. These were operated upon before we used Arfonad and Deknatel. There have been no recurrences in the last 58 cases.

9. *Pulmonary artery thrombosis with haemoptysis.*

It is noteworthy, however, that in each of the 4 cases divided, haemorrhage ensued. In my case I clamped the aorta above and below for control and this was left on for 12 minutes without any sequelae. Arfonad was also used to prevent excessive hypertension.

The average duration of stay in hospital was about 10 days.

OPERATIVE MORTALITY

We lost our 13th patient from haemorrhage at the aortic end of the ductus. This was readily controlled, but the child died of cardiac arrest. Our second death (Case No. 52) had an uneventful operation carried out for pulmonary hypertension. This child developed cardiac arrest at the end of the operation. This was due to the fact that prostigmin was given and the atropine had not yet become effective. The chest was re-opened, cardiac action was readily restored, the child living for some 36 hours before it died in status epilepticus resulting from the cerebral anoxia that had occurred before cardiac massage.

Our operative mortality is thus 2.1% (2 out of 95 cases.

OPSOMMING

Oop ductus word by ongeveer 1 uit 5,000 persone aangetref.

Vyf-en-negentig persone wat 'n operasie hiervoor ondergaan het, vorm die basis van die referaat.

Die vooruitsigte van persone wat nie behandel word nie, is funksioneel swak en die dood tree in, veral gedurende die suigelingsjare en na 40-jarige leeftyd ten gevolge van komplikasies.

Asemloosheid, lusteloosheid en asemhalingsinfeksies is gewone verskynsels. Dwerggroei word ook dikwels waargeneem. In die klassieke geval is die kliniese diagnose gebaseer op die breë-drukpolsslag, die tipiese masjineriegeruis en bevestigdende ECG en radiologiese bewys van linker-ventrikulêre hipertrofie en pulmonale volbloedigheid.

In atipiese gevalle is hartkateterisasie noodsaaklik by die vasstelling van die diagnose. Dit geld veral vir suigelinge.

Met die oog op die klein aantal ná-operatiewe sterfgevalle word chirurgie sterk aanbeveel by jong kinders, en aangeraai in alle gevalle behalwe waar 'n omgekeerde kortsluitingsirkulasie waargeneem word.

Afbinding is die chirurgiese prosedure wat toegepas is in almal behalwe 4 gevalle wat verdeel is.

This series could not have been as large as it has been without the initial help and continued co-operation for the whole of this 7 years of the Cardiac Clinic of the Johannesburg General Hospital under the aegis of Prof. G. A. Elliott.

TYPE OF OPERATION (TABLE I)

Author	*Total No. of Cases*	*Ligation*	*Operative Mortality*	*Division*	*Operative Mortality*	*Total Operative Mortality*
Ekstrom[16]	290	84%	1%	16%	16%	4%
Cade[14]	50	48	0%	4%	0%	0%
Scott[32]	273	92.3%	2.5% (under 8 mm.)	—	—	2.5%
Clagett[21]	223	82.5%	2%	—	—	—
Potts[15]	241	20%	0%	80%	0%	0%
Ekstrom[16] (Review)	1250	53%	3.6%	46%	2.4%	3%

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Dr. B. van Lingen was initially in charge of this Unit and confirmed the diagnosis in our first case. I wish to pay especial tribute to him for his constant guidance and encouragement.

Dr. M. McGregor subsequently took charge of this Clinic and throughout these 7 years both he and Dr. van Lingen have been of the greatest assistance not only in the diagnosis of these patients, but also in the management of the more complicated cases.

Dr. Josse Kaye, Head of the Radiological Department of the General Hospital, was responsible for much of the radiology in the early cases.

The low morbidity has been due to our very expert anaesthesia, most of the anaesthetics being given by Drs. Frost, Jeffes, Roberts, Meaker and Caswell.

The recent trend of operating upon the ductus in infancy has been largely due to the diagnostic acumen of Dr. Len Braudo, of Johannesburg.

Lastly, I would be failing in my duty if I did not pay tribute to the sister in charge of my Ward (Sister Duffield) and the specials (Sisters du Preez, Melville and Case) who have so willingly given of their time and energy to make these patients comfortable after surgery and to look after their physical welfare.

Case 72 was operated upon by Mr. P. R. Allison of Oxford whilst on a visit to Johannesburg, 10 cases were operated upon by my colleague, Mr. D. N. Fuller, and 3 by Mr. G. Katz, Clinical Assistant to my Unit in 1955.

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A RARE FACIAL ANOMALY

WITH A DISCUSSION OF ARHINENCEPHALIA AND AGNATHIA

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A female foetus (Bantu) of about 6 months was presented to the Department of Anatomy. No relevant information could be obtained from the mother about the period of gestation and the family history. It was still-born, showing early signs of maceration. Forceps had been applied for complete inertia in the second stage of labour.

ANATOMICAL FINDINGS

External Appearance of the Face (Figs. 1 & 2). The head was flattened in the antero-posterior

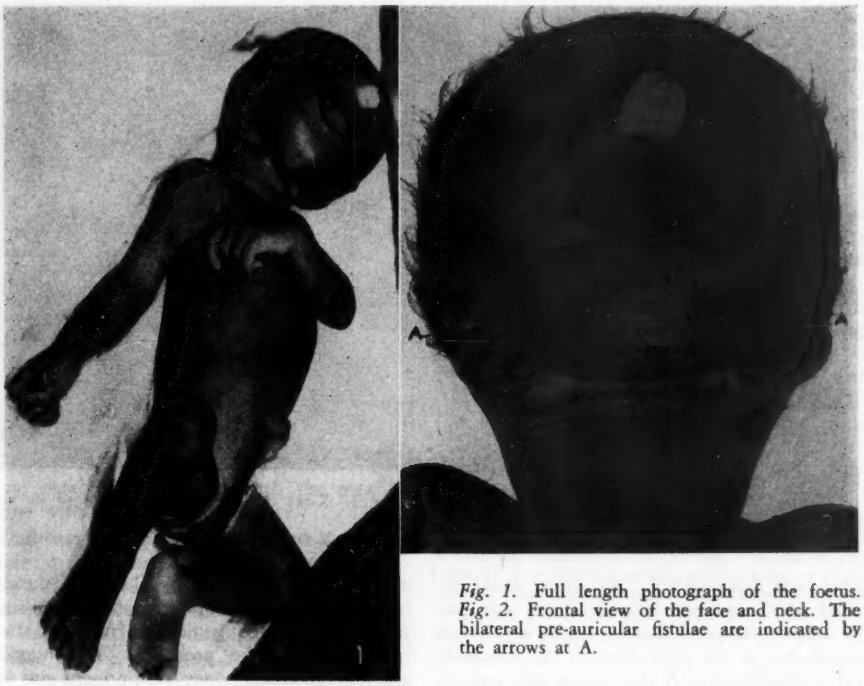

Fig. 1. Full length photograph of the foetus.
Fig. 2. Frontal view of the face and neck. The bilateral pre-auricular fistulae are indicated by the arrows at A.

diameter. A proboscis-like projection with a small blind opening was attached to the root of the forehead. Below the proboscis were two median eyes partially fused in the midline. Both eyes were enclosed by the same rudimentary eyelids and had the outward appearance of a cyclopia. A tongue-like projection guarded a small round opening which led into an abnormal oral cavity. A palate could be felt and a small pointed red object was identified as a rudimentary tongue. The ears were situated in the neck with the lobules touching in the midline. A small fistula could be seen bilaterally in front of the ears. The external appearance of the face was suggestive of complete absence of a true nose and lower jaw.

Dissection of the Soft Parts. In order to preserve the foetus for museum purposes the dissection of the soft parts was hampered and also limited by poor preservation. The scalp was incised and pulled down and the skull opened. Due to complete softening of the brain it was impossible to gather any data from it. Although discoloured, the cranial nerves and blood vessels were well preserved. The cranial nerves were systematically inspected. No trace could be found of the olfactory nerves. The frontal bones had joined in the midline, with no evidence whatsoever of a lamina cribrosa and a crista galli of the ethmoid. One small opening could be found bilaterally in the midline giving entrance to a blood vessel. The optic nerves were bilateral but very close together and left the skull through a single median foramen. The oculomotor nerves were easily identified and followed. No trace could be found of the trochlear nerves. The trigeminal nerves appeared to be normal but the mandibular division was very much reduced in size. It consisted of a small nerve bundle situated deep to the Gasserian ganglion in the position normally occupied by the motor root of the mandibular nerve. The abducens, facial, acoustic, glossopharyngeal, vagus and spinal accessory nerves could all be identified. The last of the series, the hypoglossal, could not be found.

The temporal muscle was present but ended blindly on the temporal bone. In the situation where the mandible should have articulated with the skull there was a thin ligament, 1 mm. wide and 1 cm. long. There were a few fragments (of fairly hard consistency) near its distal end. The tympanic ring was well deve-

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Fig. 3. X-ray of antero-posterior view of the skull.
Fig. 4. X-ray of the lateral view of the skull.

loped and had ossified. No trace could be found of the malleus and the incus; nor was the stapes identified, but it could have been missed due to the difficulties in the approach and its small size. A thin nerve was seen to leave the skull in the position of the foramen ovale. This nerve corresponded with the rudimentary mandibular division of the trigeminal nerve seen on the inside of the skull. The development of the hyoid bone, stylohyoid ligament and styloid process was normal. There was a muscle sheet stretching from the hyoid bone to the rudimentary oral region and filling in the intervening gap. A fairly large nerve, identified as the facial, was found bilaterally leaving the base of the skull behind the styloid process. A branch from this nerve supplied the platysma. In the midline the nerves from the two sides were completely fused, forming a horseshoe-shaped loop across the neck. No branches were given off from this loop. It presented an anatomical arrangement quite unlike any structure normally seen in this situation. None of the large salivary glands was found. Normal thyroid and thymus glands were present.

The Skull (Figs. 3 & 4). The study of the X-ray plates combined with the dissection and inspection of the parts *in situ*, yielded the following information about the skull.

The frontal bones were underdeveloped and the fusion of the right and left halves was almost complete. There was no evidence of an anterior fontanelle. The bony skeleton of the nose was completely absent. A small piece of cartilage projected from the frontal bone into the proboscis. The maxillae and the zygomatic bones were present and well developed. The temporal bone with its various parts was present. The petrous bone was seen as an outstanding landmark on the X-ray plates. As far as could be assessed, the development of the sphenoid was normal. The sella turcica and the lesser wings of the sphenoid were easily identified on the X-ray plates. The orbital cavities had developed, but were fused in the midline due to absence of the nasal skeleton.

Inspection of the thoracic and abdominal viscera revealed no congenital abnormalities.

DISCUSSION

The normal sequence of events in the development of the nose, eyes, mouth and ears will be reviewed briefly, both as regards the early moulding of the soft tissues and the subsequent development of the deeper lying cartilaginous-bony framework. With this background one has to assess in what measure the gross distortion of the face is due merely to an arrest of normal development. In a previous case report of a rare anomaly of the nose[1] it was found to be due mainly to an abnormal development of the nose.

The first outward indication of the forma-

tion of the nose[2] is the appearance during the fourth week of a pair of thickened areas of ectoderm on the frontal aspect of the head, known as the olfactory placodes. These placodes, almost as soon as they are formed, begin to sink below the general surface level so that the thickened epithelium comes to constitute the floor of the nasal pits. Surrounding the nasal pits the mesenchymal tissue proliferates rapidly so that the pits are deepened both by their own progressive invagination and by the forward growth of the surrounding tissue. The bordering elevations become the horseshoe-shaped medial and lateral nasal processes. The medial nasal processes fuse in the midline. The philtrum of the lip and the premaxilla develop from these fused medial processes. The upper lip is formed by the fused medial nasal processes (philtrum) and the maxillary processes. The lateral nasal processes fuse with the maxilla in line with the nasolacrimal duct.

This case is highly suggestive of complete failure in the anlage of the olfactory placodes. This defect in the surface ectoderm resulted in a complete agenesis of the nose (arhinia). One might go further and postulate that the agenesis of the olfactory area (surface ectoderm) resulted in secondary failure of the olfactory bulbs and nerves (neural ectoderm) (arhinencephalia). One would also expect a rudimentary development of the whole rhinencephalon, but this can only be speculation and could not be verified on the softened brain. Stupka[3] gives a description of the defects of the rhinencephalon associated with anomalies of the nose which confirms these speculations.

It is only towards the close of the second month, when the moulding of the soft tissues is well under way, that the formation of the deeper lying structures begins. The skull of a very young human embryo has a cartilaginous cranial floor (chondrocranium) with nasal capsules appearing rostrally, orbital capsules laterally and well developed otic capsules on the sides further caudally. In this case the nasal capsule failed to develop. The bone of the nasal capsule is the ethmoid which, with the sphenoid and occipital bones, are the three main bones of the base of the skull. The ethmoid of the young chondrocranium consists of a median and paired lateral parts. The medial portion extends to the tip of the developing nose. Its more rostral part persists with little alteration as the cartilaginous part of the nasal septum. Its main portion gives rise to the perpendicular ethmoid plate. The lateral portion gives rise to the ethmoidal air cells and superior and middle turbinates. Between the medial and lateral ethmoids the olfactory nerves go through the cartilage which becomes ossified round the olfactory nerves and is known as the cribiform plate.

During development of the face, the eyes undergo a striking change in their relative position (Figs. 5A, 5B). At 6 weeks the eyes are still far to either side of the head, almost like the eyes of a fish. As the facial structures grow, the eyes are carried forward in the head. The primordia of the nose having failed to develop, the eyes were carried too far forward, virtually into the median position normally occupied by the nose (Figs. 6A, 6B). The proboscis-like nose is probably the result of an abnormal overgrowth of the soft tissues of the frontal process which would normally form the bridge of the nose. Patten states:

> 'The distortion that results in cyclopia usually involves other structures in the front of the head. A common concomitant of cyclopia is a nose in the form of a cylindrical proboscis-like mass of flesh pendent from the nasal region or forehead'.

In this particular case there were two optic nerves and bilateral innervation of the muscles of the eyes. The absence of the trochlear nerves might be due to a failure of the superior oblique muscles to develop due to partial fusion of the eyes in the midline.

The development of the lower jaw goes back to a very early age of embryonic life. The most conspicuous landmarks at the fourth week are the stomodaeal depression and the mandibular arch. The mandibular arch is covered with surface ectoderm and has a core of mesodermal tissue. On the inside it is related to the oral cavity. Associated with the chondrocranium is the visceral skeleton. The mandibular or first gill-arch skeleton is represented by Meckel's cartilage. The second, hyoid arch, by Reichert's cartilage. The third, fourth and fifth are concerned with the laryngeal cartilages.

The following structures are said to develop in relation to the mandibular arch:

The mandible, spheno-mandibular ligament, malleus, incus and the muscles of mastication innervated by the mandibular division of the trigeminal nerve.

Richany[4] gives a detailed description of the ossification of the mandible which takes place in membrane and starts at the level of the future mental foramen. Arey[5] describes the first branchial arch forking into a rostral maxillary and caudal mandibular process and there-

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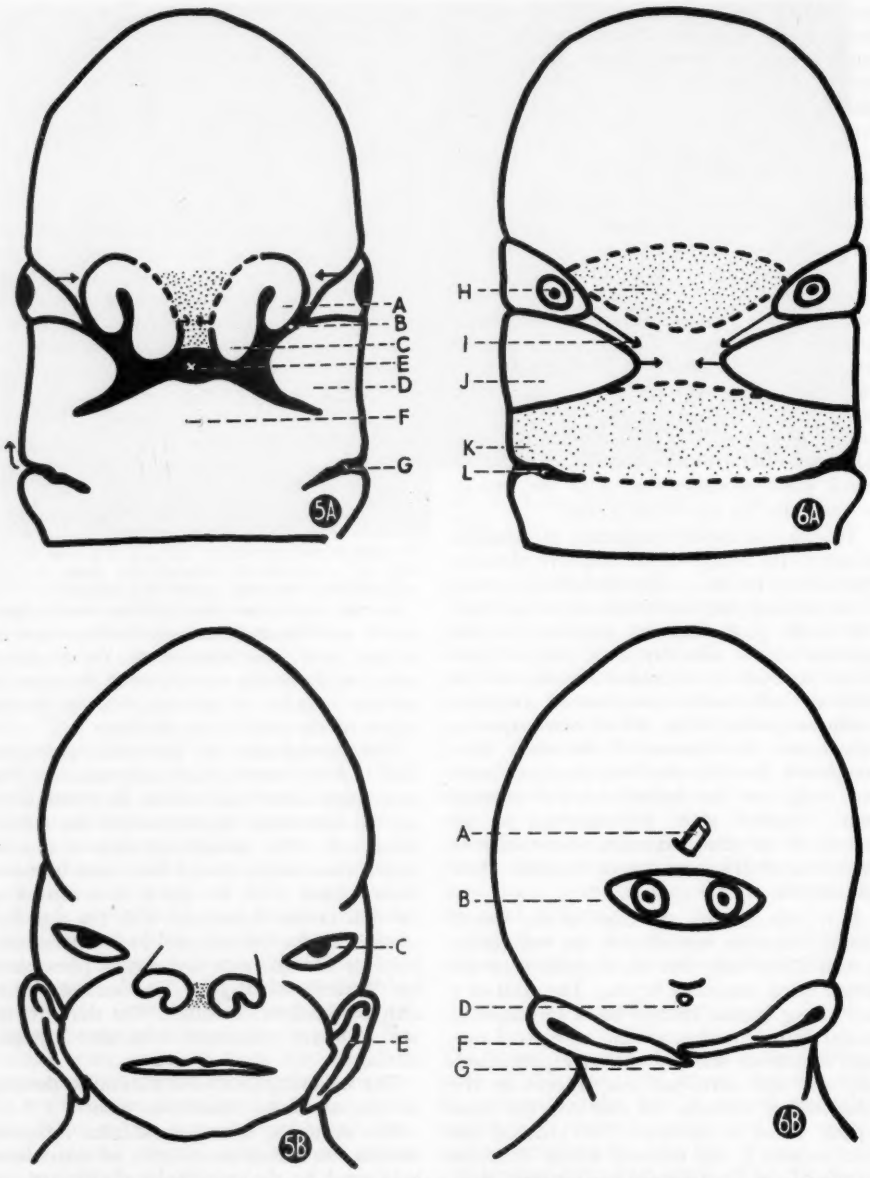

Figs. 5A, 5B. Normal development of the face at 6 and 8 weeks to demonstrate the relative change in position of the eyes and external ears. (*Redrawn from* B. M. Patten's *Human Embryology*).

Figs. 6A, 6B. Abnormal rotation of the eyes and arrest of the external ears in the neck due to agenesis of the olfactory pits, the nasal processes and the mandibular arch.

Figs. 5A, 6A.

A: Lateral nasal process.
B: Naso-optic furrow.
C: Medial nasal process.
D: Maxillary process.
E: Stomodaeum.
F: Mandible.

(*Legend continued on p. 403*)

fore regards it as a branchial arch derivative. This does not seem feasible when considering the anatomical data presented by this case. Here there is an agenesis of the mandibular arch but a normal development of the maxillary process. This fits in with Keen's findings[6] in a case of agnathia, where he came to the conclusion that the maxillary process did not take origin from the dorsal part of the mandibular arches. Starck[7] describes the maxilla as having a separate origin from the mesenchyme in the region of the nasal capsule and is therefore a separate entity. It was noted that the mandibular nerve was not completely absent. The slender nerve present was suggestive of the motor root of the mandibular nerve.

A well developed temporal muscle was found in relation to the temporal bone and ending blindly on it. Due to the poor preservation of the soft tissues, detailed data could not be gathered about the other muscles of mastication. There was a definite muscle layer present which could be seen stretching from the hyoid bone and forming the so-called floor of the maldeveloped mouth. It resembled an abnormal mylohyoid muscle and probably developed with the hyoid arch.

With these incomplete data one cannot come to very definite conclusions, but it is suggestive that the muscles of mastication are not all primarily developed in association with the mandible and that certain muscles migrate and become attached to it secondarily. This would account for the presence of the motor part of the mandibular root of the mandibular nerve but the complete absence of the sensory part, due to agenesis of the ectodermal covering of the mandibular arch. One might postulate a primary defect in the surface ectoderm which profoundly influenced the subsequent sequence of events in the underlying mesenchymal tissue and neural ectoderm including the peripheral nerves which normally supply it. Failure of the mandibular arch resulted in gross malformation of the mouth, the tongue and the salivary glands.

The lateral lingual swellings, a mandibular arch derivative, failed to develop. This normally gives rise to the mucosal covering of the bulk of the tongue, the largest part anterior to the sulcus terminalis. Its sensory nerve comes from the lingual branch of the mandibular nerve. The mucous lining of the body of the tongue having failed to develop, the muscles of the tongue, which are thought to be derived from the occipital myotomes, also failed to develop. This might explain the absence of the hypoglossal nerve. The entodermal structures, thyroid and thymus, which develop from the second, third and fourth pharyngeal pouches, were quite normal in size and position.

Another sequela of the agnathia is the embryonic position of the external ears, i.e. in the position of the hyo-mandibular cleft in the neck. Development of the lower jaw normally forces the ears round the side of the head. One has to find some explanation for the abnormal but well developed facial nerve. The development of the hyoid arch was normal as far as could be assessed. The muscles of facial expression and platysma are regarded as mesodermal derivatives of this arch. In the neck a well developed platysma was found. The muscles of facial expression are concentrated around the orifices of the face. One might suggest that due to the gross abnormalities of the nose, eyes, mouth and ears these muscles failed to develop, being very closely associated with the skin covering these structures. It is probable, therefore, that the development of the muscles of facial expression is determined to a very large extent by the surface ectoderm of the face and neck and the facial nerve associated with the hyoid arch migrates to supply these muscles.

The main defect in this case seems to be a developmental arrest at a very early stage of embryonic life of the nasal area and the mandibular arch. The anatomical findings are suggestive of a primary defect in the surface ectoderm of these areas which profoundly influenced the development of the mesenchymal tissue, the rhinencephalon and several of the cranial nerves. Blechschmidt[8] showed that the embryonic skin influenced the developing skeleton.

G: Hyomandibular cleft.
H: Agenesis of the olfactory pits and nasal processes.
I: Abnormal rotation of eyes.

Figs. 5B, 6B.
A: Proboscis-like nose.
B: Abnormal rotation of eyes. Partial fusion.
C: Normal rotation of the eye.
D: Rudimentary button mouth.
E, F: External ear.
G: Non-rotation of ears.
J: Maxillary process.
K: Agenesis of the mandibular arch.
L: Hyomandibular cleft.

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Little or nothing is known about the causation of arhinencephalia and agnathia. The many and various etiological factors cited for congenital abnormalities of the face and anomalies in general are also mentioned here as possible causal factors.

SUMMARY

A gross malformation of the face is described, presenting a proboscis-like nose, median eyes, button mouth and ears situated in the embryonic position in the neck.

The primary defect seems to be the agenesis of the nasal region (arhinencephalia) and mandibular arch (agnathia).

As a sequela of the agenesis of the nose, the eyes migrated too far towards the midline (cyclopia).

The agnathia had as a result the rudimentary mouth and persistence of the embryonic position of the ears.

The anomalies in this case are mainly due to an arrest in development at a very early age of embryonic life rather than an abnormal development.

It is suggested that the primary defect might have been in the surface ectoderm. The causation of this defect is unknown.

OPSOMMING

'n Uitgebreide misvorming van die gesig, met die uiterlike beeld van 'n slurpagtige neus, mediaanlyn oë, rudimentêre mond en ore in die embrionale posisie in die nek, word beskryf.

Die primêre afwyking skyn die agenese van die neusgebied (arhinencephalia) en mandibulare kiefboog (agnathia) te wees.

As gevolg van die agenese van die neus het die oë te veel roteer na die mediaanlyn (cyclopia); as gevolg van die agnathia, die rudimentêre mond en die ontwikkeling van die ore in die embrionale posisie.

Die anomalieë in hierdie geval is in hoofsaak te wyte aan 'n onderbreking en nie ontwikkeling in die embrionale geskiedenis.

'n Tentatiewe voorstel word gemaak, dat die afwykinge as gevolg van 'n primêre swakheid in die anlage van die oppervlakte ectoderm mag wees, die oorsake waarvan onbekend is.

I wish to thank Dr. I. J. Louw of Bethal for preserving and sending the foetus to the Department of Anatomy with excellent photographs. I am also indebted to the X-ray Department, Pretoria General Hospital, for the X-ray photographs of the foetal skull.

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THE INITIATION OF BREAST FEEDING IN THE PUERPERIUM

FOUNDATIONS OF THE FEEDING RELATIONSHIP

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When breast feeding is successfully initiated, the prenatal biological relationship between mother and foetus becomes both a biological and social one after birth. Breast feeding is so significant in the overall mother-child relationship of early infancy that the term 'feeding relationship' is preferred to that of infant feeding.

The newly born baby's complete dependence on others for the fulfilment of his needs is reflected in the close mother-child relationship characteristic of most societies during the early period of infancy. Thus, even among those who do not put the baby to the breast in the early days after birth, mother and baby are closely associated in the period of post-natal confinement.

One of the most striking changes that has occurred in Western European and North American cultures has been the degree to which mothers and babies have been separated immediately after birth. This is no doubt related to the increasing numbers of women who have their babies in hospital. Until very recently the most effective maternity units in hospitals were generally considered to be those in which

there were separate facilities for mothers and babies. The result is that in many hospitals babies are cared for in special nurseries while mothers are separately cared for in maternity wards. In these conditions the babies are brought to their mothers at feeding times, thus limiting contact between them to very short periods. During the greatest part of the day the baby is in its crib in the nursery, supervised by nursing staff.

Another feature of the change which has occurred in these societies has been the rapid extension of a belief in fixed-time feeding with the result that it has become their customary practice. The deep roots which this belief had taken is indicated by the following quotation from *Modern Methods of Feeding in Infancy and Childhood* (Patterson and Smith, 1939):

'When the secretion of milk is established, absolute regularity of feeding must be insisted on from the start. There is still a difference of opinion as to whether three-hourly or four-hourly feeding gives the best results.'

It is noteworthy that no mention is made as to possible differences of opinion on the question of time feeding itself.

The dogma that so readily emerges from such beliefs is well illustrated in the following extract from *A Textbook of Gynaecology with Obstetrics* (Fairbairn[3]).

'A baby differs from the suckling animal or from the baby of the uncivilized savage in that, as he grows up, his mealtime, sleep-time, play-time, and work-time will be determined by social circumstances. The careless, shiftless and ignorant mother whose child is brought up without method, and given the breast whenever he cries for it, is injuring both the health and character of her child. Not only is he likely to have disturbed digestion and irregularities in the action of the bowels, but he is acquiring the slipshod ways of his parents, and without discipline in self-control, he grows up self-willed and unable to adapt to our customs, and is neither physically nor morally a credit to the race. This latter aspect is one that is often lost sight of, but should be put before every mother who is capable of appreciating the importance of this early discipline and of its effects on the future upbringing of the child.'

Few would disagree with the serious effects of careless and shiftless mothering, but giving the baby the breast when he cries for it can hardly be a criterion for the diagnosis of maternal inefficiency.

Increasing doubts are being expressed about the desirability of these comparatively recent changes, and the efforts are now being made to modify them by encouraging closer and more flexible mother-child relationships. Before considering these developments, it might be well to review some of the features of present-day care of infants in this country, more especially that of the Zulu, whom we of the Institute of Family and Community Health have had an opportunity of studying during the past 17 years.

One of the earliest clinical impressions that my wife and I had when we started medical practice among the rural Zulu of Polela, was the apparently high incidence of diarrhoea in early infancy. As all the infants seen had been born at home, the condition was not thought to be related to that which has been reported in newborns in hospital nurseries.

Investigation of home management revealed the following relevant findings. Mothers did not put their babies to the breast until colostrum had been replaced by the free flow of milk. This varied from the third day to as late as the fifth day. The infant was fed, by bottle usually, on boiled water and incumbe, a home-made cereal water drink, made either from maize or sorghum. In addition, immediately after birth the baby was given an enema to remove the meconium. This cleansing process was continued once or twice daily for the first week, and remained a feature of infant management, and later in the care of the sick at whatever age. It was considered that these practices, cereal feeding and frequent enemas, might well be the cause of the neonatal diarrhoea which was apparently so common.

While the infant was receiving this type of care, the mother's breasts were massaged and warm cloths were applied. This milking-off process succeeded in preventing over-distension and tension of the breasts and it was exceptionally rare for mothers to have any difficulty in initiating and maintaining satisfactory breast feeding. A further factor of significance to the present discussion is the extent to which young girls were encouraged to manipulate their breasts and nipples. The stated objective was aesthetic, but the effect of drawing out the nipple from the time of early development of the breasts during puberty must have a considerable effect on the incidence of retracted nipples and on the hardening of the nipples. Cracked and tender nipples were rarely seen in practice.

During the early days after birth, mother and baby were confined together in a hut and a very close physical relationship was maintained. Once breast feeding was initiated, an indulgent feeding relationship was established from the beginning. Mothers responded to babies' demands as they arose, and it was of interest to watch the experienced mother's

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response to her baby's crying and movements that meant the baby wanted something. Not all demands were met by giving the baby the breast to suck. Holding the baby up, or rocking it and talking or singing to it, were often enough to soothe it back to sleep or quiet. Never was there an obsessional thrusting of the baby to the breast at the slightest cry. The outstanding feature was that of a confident mother or, in the case of a young mother with her first baby, a confident grandmother guiding the process, establishing an easy feeding relationship with baby. I prefer to use the term 'adjusted feeding relationship' to describe this, instead of 'self-demand feeding', as the latter refers only to the baby. The process as observed in Polela, and among the Africans of Durban, involves a mother who often feels that her baby needs the breast even before he demands it by crying.

The fact is that the successful initiation of lactation is associated with a very high incidence of breast feeding in the later months of infancy, as indicated by the following figures of the incidence of breast feeding in African babies of Polela and Durban at different ages to the end of the first year:

	At 1 *Month*	3 *Months*	6 *Months*	9 *Months*	1 *Year*
Durban	99.4	90.7	82.2	71.7	69.4
Polela	99.4	99.2	96.6	96.3	84.4

The more rapid decrease in the incidence of breast feeding of the Durban babies after the first month is indicative of an important change taking place among urban Africans. It may well be an early sign of the decline in breast feeding which has been very noticeable among European and American peoples as they became affected by industrial urban living conditions.

Zulu mothers of Durban who were discussing the question of breast feeding referred to differences between themselves and European mothers, for whom some of them had worked as domestic servants, in terms which have relevance to the present discussion.

'It is not true to say that European mothers (or most of them) have not sufficient breast milk, or that it dries up because they want it to do so. They feed their babies according to fixed times, and breasts normally do not work according to the clock. Since they force the breasts to work at the times they want them to, they meet such results as milk drying off or decreasing until they say they cannot feed their babies because they have no milk. Milk is stimulated by the baby sucking, and sucking each time the breasts are full, so that by trying to empty them the milk flow is increased. So the process of emptying and filling continues. The Europeans feed by clock with intentions not mainly to nurse, but to keep themselves up with daily enjoyments, which otherwise would be denied them if they had to feed their babies each time they wanted the breast.

Secondly, European mothers do not sleep with their babies. A baby grows from birth with a feeling of isolation and a knowledge that it will not have any night feeds. Though a baby might respond well to a routine carried on from birth, the breasts do not since they become starved of stimulus and they react by decreasing the outflow of milk.'

Another added:

'It is also a shame amongst many European mothers to feed in public, even in the presence of another female. One mother for whom I worked, when she had to breast feed, would close the door, draw the curtains, sit down comfortably, taking time before she started feeding. If anybody came into the house she would give me the baby to look after and she would attend to the guest. That baby was not properly fed; it did not have what it should have because she was ashamed of feeding it in the presence of people. A women's pride lies in the successful nursing she has given.'

Whether or not these are among the reasons for the differences in the prevalence of breast feeding by African and European mothers of this country, they do reflect some of the differences in the feeding relationship between mothers and babies of the two groups as seen by African women. All the women in the discussion were agreed that the successful initiation of lactation and its maintenance were associated with the close relationship of mother and baby, continuing from birth through infancy. In so doing, some of them spoke disparagingly of the modern trend in their own community whereby some women wish to be with their husbands rather than with their babies. The fulfilment of the maternal role was regarded as the highest status which a woman could attain; her success or otherwise as a wife was felt to be determined more by her success as a mother than by any other roles fulfilled as wife.

The decline in breast feeding in various countries has been one of the factors stimulating a re-appraisal of feeding habits. In particular, attention is being directed to the initiation of breast feeding and the over-all mother-baby relationship during the puerperium. It is of interest to note that two of the features of the approach now being made to reduce the high incidence of lactation failure are of the kind which we noted in Polela women. They are concerned (1) with attention to the breast to prevent over-distension and to prepare the nipples and (2) with encouraging a close mother-baby relationship associated with a flexible schedule of feeding in which baby's

demand is taken into consideration. The results of these approaches will be considered here.

PREPARATION OF THE BREAST

Waller[10] drew attention to the importance of over-distension of the breast during the puerperium by a controlled study of primiparae in which the prevention of overdistension was associated with considerable success in the initiation and maintenance of lactation; 83% of those treated were successfully breast feeding their babies at 6 months of age in contrast to 42% in the control group.

Briefly stated, his method includes careful ante- and post-natal attention to the breasts, with the object of preventing increased milk tension in the breast. This he believes to be the main cause of difficulty in starting breast feeding. Anatomical defects of the nipples and terminal ducts are considered to be responsible for the frequency of 'high milk pressure' at the outset.

The antenatal care includes careful inspection of the nipples. Not only are retracted and inverted nipples considered of significance but the range of protraction is stressed. On this will depend the degree to which the baby can draw the nipple well into its mouth. Glass shields with a central opening are firmly applied to such cases, the nipple being gradually pressed through the opening. The majority of the cases responded satisfactorily to this treatment. A second feature of Waller's measures is the antenatal expression of colostrum during the last 3 months of pregnancy. This was thought to encourage the free flow of milk after birth, and at the same time provided the mother with the skill she required for post-natal manual expression.

The post-natal care again stressed the need for careful examination of the breasts to ensure early diagnosis of increased tension. He recognizes 4 degrees of this condition:

i. *Normal increase,* with free outflow of milk in which manual removal of milk is not needed.

ii. *Overload,* greater than (i) but readily reduced by manual expression.

iii. *Engorgement,* often associated with observable oedema of the skin. The engorgement makes the nipples less protractile. It can be reduced by manual expression, and he withholds baby from the breast until it has subsided.

iv. *Engorgement with obstruction.* This is the extreme degree with no outflow of milk. The breasts are raised and firmly supported. No attempt at feeding is allowed and manual expression is begun as soon as any outflow can be obtained. Feeding is begun when the condition has subsided, and is associated with manual removal of excess milk both before and after meals. Stilboestrol is used in such cases.

A trial of Waller's methods has been carried out by the Departments of Obstetrics and of Child Health at Guy's Hospital.[1] Their trial included control and experimental groups of primiparous women delivered in hospital as well as in their homes. The results of the cases delivered in hospital were confirmatory of Waller's findings. The numbers of women who successfully fed their babies for 6 months was nearly double in the 'Waller' group than in the controls (51% as against 26%), engorgement of Waller's third and fourth degrees was very much less common (15% and 37% respectively) and there were fewer cases with cracked nipples (3.6% and 7.8% respectively). They also confirm the value of the glass shield in the treatment of abnormal nipples.

The results in the cases delivered on district were different. No significant difference in the incidence of breast feeding between the 'Waller' group and the controls was noted, and both were very much lower than the Waller group delivered in hospital:

District 'Waller' Group — 22%.
Control — 31%.
Hospital 'Waller' Group — 51%.

They consider that this indicates that it is the supervision in the lying-in period that counts, and conclude that 'if this is so, then the antenatal massage and expression may be considered simply as preparation for the use of these methods for removing surplus milk after feeds in the puerperium'. This conclusion receives some support from our observation of Zulu mothers in Polela. Expression of milk takes place only after the birth of the baby, successfully preventing engorgement, without the prenatal expression of colostrum. Attention to the nipple, on the other hand, begins at puberty.

Another difference between their hospital cases and those delivered on district is of interest. Ten per cent of hospital babies were having complementary bottle feeds in addition to the breast at the end of the lying-in period, as compared with none of the district babies. They consider that this difference is not merely because milk is more readily available is hospital, but that it is the result of a number of interrelated factors, including the following:

(*a*) In hospital a baby is handled by a larger number of different people, with varying experience. In difficult cases, the mother will receive help and advice from the nursing staff, and because of changes of staff during day and night the advice may vary.

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(*b*) The baby may be disturbed by going from a nursery to a ward at a different temperature.

(*c*) There are more difficult deliveries among hospital cases and the babies of these cases may have more difficulty in feeding.

(*d*) Babies in the hospital were fed at regular intervals by the clock. District cases would probably be on a less rigid schedule of feeding.

Three of the factors which they consider may have been important in explaining the frequency of complementary milk feeds in hospital are relevant in our appreciation of the adjusted feeding relationship, which we have noted as an important feature of the close association between Zulu mothers and their babies. Attempts to re-establish this close association in other communities have led to the 'rooming-in' of mother and baby, as well as the re-introduction of a flexible feeding relationship.

The part which the baby can play in preventing engorgement of the breasts and sore nipples is being further investigated in relation to the question of the effect of flexible feeding regimes. It is well known that the baby's sucking or milking-off are stimulants for the secretion of milk. Of equal importance is it that the milk shall flow freely and thus be made available. The virtue of Waller's studies is their stress on the importance of the expulsion of the milk which has already been secreted. Waller[9] and later Newton and Newton[6, 7] have shown that the mechanism involved in women is similar to that known as the let-down reflex in animals. Sucking is probably the primary stimulus for this reflex, but it is readily conditioned by such factors as the sight or thought of the baby and the time of feeding.

If sucking by the baby is so important a stimulus to this reflex and hence to the free flow of milk, it might be expected that satisfactory breast feeding would achieve the same desirable end as does manual expression. A trial of 'self-demand' feeding regime as compared with rigid time-feeding in Sheffield, England, indicates that this is so.[4] The results of this carefully controlled study showed that there were more than double the number of cases of over-distension of the breast and soreness of the nipples in the 'rigid' group than in the 'demand' group—of the 'rigid' group, 34.0% developed over-distension and 27.4% sore nipples, compared with 16.9% and 12.9% respectively of the 'demand' group. Furthermore, apart from these factors of over-distension and sore nipples, the demand group had a higher incidence of breast feeding. Thus in cases where there was no over-distension, the incidence of breast feeding on discharge from hospital and at 1 month indicated the following differences:

	'Rigid' Group	*'Demand' Group*
On discharge	84.4%	93.1%
At 1 month	55.9%	80.2%

This study, and that of Nyhan and Wessel[8] from the Yale Rooming-In Project, Yale University School of Medicine, present evidence of the difference in weight growth of babies fed on different regimes. There was less loss of weight during the immediate postnatal period, and a more rapid return to birth weight, in the babies fed on a flexible regime than those time-fed. This is probably mainly related to the difference in quantity of milk taken by the different groups.

It is obvious that if an adjusted, flexible feeding relationship is to be encouraged from the outset (and available evidence suggests that this is desirable) arrangements must be made in hospitals for mothers and babies to be together. In hospitals where this has been done, reports are encouraging. Of particular interest are those who report that the 'nurses found that a self-demand schedule was a great deal easier for them'[4] and that there is a considerable saving in nursing time and hence in staff requirements.[5]

CONCLUSION

The prevention of post-partum engorgement is both possible and important. Its importance is that it is associated with failure to establish satisfactory lactation in that it is a painful condition which can go on to cracked nipples and breast abscess and it interferes with the outflow of milk. As such it is a most important factor in disturbing the development of a feeding relationship, which is the early foundation of the relationship between mother and child.

The traditional methods of early feeding among the Zulu, as indicated by our observation of Polela mothers, includes some of the most important features of recent efforts to re-establish breast feeding in Western societies.

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Among these are care and preparation of the nipples, and the prevention of engorgement by post-partum manual expression of colostrum and milk followed by a flexible feeding regime in which the baby's demand and the mother's response are adjusted and individualized. It might well be that putting the baby to the breast as soon as it shows the desire would be an advance on the Polela practice of waiting until the milk flow is established. This has been the practice of the Institute of Family and Community Health for some years. The response of urban African and Indian women has been satisfactory, and in the rural community of Polela, Cassel[2] had some interesting results. The average loss of weight in 62 full-term healthy babies was only 2.01% (the 33 boys losing an average of 3.33 oz. and the 29 girls 1.61 oz.); 56% had regained their birth weight by the third post-natal day and a further 40% (total 96%) regained or exceeded their birth weights by the end of the first post-natal week. Whether this limited post-natal loss of weight is of significance to the health of infants remains to be determined. In this particular case, viz. Polela, it is a decided advance on the previous custom of cereal feeding during the prelactation period, and neonatal diarrhoea of the kind that used to occur decreased as this habit was changed.

Early post-natal breast feeding on a flexible schedule might therefore reduce the need for manual expression. A combination of the two, varied according to individual need, would constitute a significant advance in the prevention of post-partum breast engorgement and the promotion of an easier initiation of breast feeding.

OPSOMMING

Die voorkoming van kongestie na 'n bevalling is nie alleen moontlik nie maar ook van groot belang. Die belangrikheid daarvan moet toegeskryf word aan die feit dat dit geassosieer is met die versuim om bevredigende melkafskeiding teweeg te bring. Dit is 'n pynlike toestand wat kan uitloop op gebarste tepels en borsswere, en dit belemmer die uitvloeiing van die melk. As sodanig is dit 'n uiters belangrike faktor in die versteuring van die voedingsverhouding, die eerste grondslag van die verhouding tussen moeder en kind.

Die tradisionele metodes van vroeë voeding onder die Zoeloes, soos aangedui deur ons waarneming van Polela-moeders, behels 'n hele paar van die belangrikste kenmerke van die jongste pogings om borsvoeding in westerse gemeenskappe te herstel. Onder hulle is daar die versorging en voorbereiding van die tepels, en die voorkoming van kongestie ná die bevalling deur die uitdrukking met die hand van kolostrum en melk, gevolg deur 'n buigbare voedingsregime waarin die babetjie se vereistes en die moeder se reaksie aangepas en geïndividualiseer word. Dit kan wees dat die gewoonte om die kind aan die bors te sit sodra hy die begeerte toon, 'n verbetering is op die Polela-gewoonte om te wag totdat die melk bestendig vloei. Dit is reeds 'n hele paar jaar lank die gebruik in die Instituut vir Gesins- en Gemeenskapsgesondheid. Die reaksie van stedelike naturelle- en Indiër-vroue was bevredigend, en in die plattelandse gemeenskap Polela het Cassel[2] 'n aantal interessante resultate behaal. Die gemiddelde verlies van gewig by 62 babetjies wat na die normale tyd gebore is, was slegs 2.01% (die 33 seuntjies het gemiddeld 3.33 ons ligter geword en die 29 dogtertjies 1.61 ons); 56% het hul geboortegewig op die derde dag na geboorte herwin, en 'n verdere 40% (totaal 96%) het hul geboortegewig teen die einde van die eerste week na geboorte herwin of oorskry. Of hierdie beperkte na-geboortelike gewigsverlies van belang vir die gesondheid van suigelinge is, sal nog vasgestel moet word. In hierdie besondere geval, naamlik Polela, was dit 'n besliste verbetering op die vroeër gewoonte om die babetjie graankos te gee gedurende die tydperk vóór melkafskeiding, en neogeboortelike diarree van die soort wat tevore voorgekom het, het verminder toe hierdie gewoonte afgeskaf is.

Vroeë na-geboortelike borsvoeding volgens 'n buigbare skedule kan derhalwe die noodsaaklikheid vir uitdrukking van die melk met die hand verminder. 'n Samestelling van die twee, gewysig ooreenkomstig individuele behoeftes, sou dus 'n betekenisvolle vooruitgang wees in die pogings om borskongestie na bevalling te voorkom en om die gemakliker instelling van borsvoeding in die hand te werk.

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XUM

CEREBRAL PALSY HABILITATION IN SOUTH AFRICA

S. P. FREELAND*

In common with many other countries, South Africa has seen a tremendous growth of interest in the problem of cerebral palsy during the last 10 years. Inspired by the new hope offered to these handicapped people through the work of people like Winthrop Phelps, Temple Fay, Earl Carlson, the Bobaths and many others, parents have come together and insisted that the best available treatment be made available to their children in this country. As a result, various projects are now in existence (treatment centres, schools, parents' groups, committees and associations) in the main centres of South Africa.

The first of these to be set up was the Johannesburg School and Treatment Centre for Cerebral Palsied Children (originally known as the Forest Town School for Spastics). This was established in 1948 by the Transvaal Association for the Care of Cerebral Palsy, and began in a Church Hall with 6 children in the charge of a speech therapist. During the 9 years of its existence it has developed into a modern school and treatment centre comparable with some of the best in leading countries overseas, with 100 scholars and a staff of nearly 30—medical, therapeutic, educational, etc. In addition, there are nearly 200 children attending the outpatients' clinic more or less regularly, and a large panel of specialists whose services are given as required from time to time.

Encouraged by the success of the Johannesburg experiment, similar institutions have been established in Pretoria, Cape Town and Port Elizabeth. All 4 are now being subsidized by the Union Government under the Special Schools Act, and receive grants which cover just over one half of their annual expenditure. These centres keep in touch with the latest developments in the field of cerebral palsy habilitation, and use various methods and techniques of treatment. At present they are day centres only, but the Johannesburg School is now in the process of establishing a special hostel for children from country districts. Similar facilities will soon become available at Cape Town, whilst in Pretoria, a completely new school and hostel is to be built.

Cerebral Palsy Associations or Divisions have now been established in the Southern and Northern Transvaal, the Cape and the Eastern Province. It is hoped that a similar body will be set up in Natal, and special committees for cerebral palsy work exist in East London, Bloemfontein, Kimberley and Windhoek. All these organizations are tackling the problem of cerebral palsy in their own areas. In Durban, for instance, there is a special class for cerebral palsied children as well as a parents' study group. In Bloemfontein plans are being initiated to set up a reassessment clinic for the careful examination and testing of all known cases in the area. Excellent work of an exploratory character has been done in East London, whilst plans are afoot in South West Africa to build a suitable centre for the treatment of the cerebral palsied. In Johannesburg and at Edendale in Natal, preparations are going ahead for the establishment of non-European clinics, whilst just outside Pietermaritzburg a number of cerebral palsied children are being given treatment and schooling at Uplands.

All this work is being co-ordinated by the Cerebral Palsy Division of the National Council for the Care of Cripples in South Africa, to which the various bodies already referred to are affiliated. The Council, as a result, has become (through its Cerebral Palsy Division) the national body in South Africa for this particular problem in all its aspects. It has worked in this field for a number of years, and 3 of the 4 schools and treatment centres now recognized by the Government were initiated by the Council or its Regional Associations. The original Cerebral Palsy Committee of the National Council, which for some years gave oversight to cerebral palsy work all over the country, was expanded into a special department at the Annual General Meeting of the National Council in October 1955.

The following are some of the aims of the Division:

To work for complete unity in the field of cerebral palsy habilitation in South Africa;

To establish facilities for the diagnosis and treatment of cerebral palsy in the main centres of the Union;

* Rev. S. P. Freeland is Chairman of the National Council for the Care of Cripples' Cerebral Palsy Division, and Vice-Chairman of the Johannesburg School and Treatment Centre for Cerebral Palsied Children.

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To educate the public about what this problem is and how it can be effectively dealt with;

To give special attention to the care and treatment of those regarded as ineducable;

To train personnel (doctors, therapists, teachers, etc.), in order to staff adequately the various institutions set up;

To compile a national register of all known cases;

To give attention to the problem as it affects non-Europeans;

To embark upon clinical and educational research;

To establish hostels in large centres; and

To provide vocational guidance and training, and subsequent employment, for the adult cerebral palsied.

One of the first actions of the Division was to give assistance to the Eastern Province Cripple Care Society in the establishment of the Cerebral Palsy School at Port Elizabeth, mentioned earlier in this article. The first issue of *The S.A. Cerebral Palsy Journal* was also published, and has continued as the quarterly organ of the Division. It has already proved to be a most valuable medium of information and news for those working among the cerebral palsied. Another most successful venture of the Division was the holding of a 3-day Vacational Course on the Education of Cerebral Palsied Children in October 1956, when nearly 100 persons enrolled. The addresses given at this course were subsequently published.

This article would not be complete without mention of a small but useful contribution to the programme of habilitation for South Africa's cerebral palsied, viz. the clinic established a year ago in Rosettenville, Johannesburg. No figures are available about the actual work being done, as the clinic is being run by an independent organization, known as the United Cerebral Palsy Association, affiliated to an American body of the same name. It is known, however, that at this centre, diagnosis and treatment are given for the ineducable child, and that a team of specialists gives their assistance in honorary capacities. It was hoped that this valuable piece of work would come under the aegis of the Division's national network of centres and organizations, but up to the present the sponsors have preferred to work independently.

Interest in the work, carried on among the cerebral palsied in many lands, is being further quickened in view of the first World Cerebral Palsy Conference to be held in July this year in London and organized by the International Society for the Welfare of Cripples. Delegates will be present from all over the world, including 3 from South Africa, and it is expected that the discussions which will take place will lead to further developments in the field of cerebral palsy habilitation.

OPSOMMING

Die skrywer verstrek 'n oorsig van die werk wat gedoen is om die versorging van serebraalverlamde kinders in hierdie land te verbeter, en vestig die aandag op die oogmerke van die Nasionale Raad vir die Versorging van Kreupeles in Suid-Afrika.

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REVIEWS OF BOOKS

AN ATLAS OF CLINICAL ENDOCRINOLOGY

Atlas of Clinical Endocrinology. By H. Lisser and R. F. Escamilla. 1957. 1st ed. (476 pp. with 148 plates, 3 in colour. £8 0s. 6d.). St. Louis: The C. V. Mosby Company.

This *Atlas* is a remarkable and timely contribution to clinical endocrinology. It is more or less equally divided into text and illustrations and therefore constitutes what is virtually a bedside guide to the practitioner. Almost every endocrine disorder is strikingly illustrated with informative clinical photographs.

The *Atlas* compresses an enormous amount of clinical information into an extremely concise compass. It provides a valuable guide to the practitioner in respect of special laboratory investigations which may be helpful in evaluating symptoms and signs of what can be extremely complicated cases.

The authors are well known as distinguished practitioners in the field of clinical endocrinology. Their names have become associated eponymously (in the Escamilla-Lisser syndrome) with a constellation of symptoms in which the external appearances may not be suggestive of an under-functioning thyroid gland, but in which investigation reveals many 'internal' manifestations of myxoedema.

The general practitioner will find the *Atlas* a particularly valuable means of classifying many of the commonly encountered endocrinopathies involving such features as obesity, hirsutism and gradations of sexual abnormality.

In the discussion of Simmonds' disease, more attention should be focused on the absence of cachexia, as is well brought out in Sheehan's syndrome of post-partum necrosis of the anterior lobe of the pituitary gland. Extreme emaciation is a feature of anorexia nervosa rather than of destruction of the hypophysis.

The *Atlas* also gives an instructive and succinct account of a fairly new endocrinopathy (Conn's disease or primary aldosteronism) associated with the excretion of excessive amounts of this hormone in the urine of oedematous nephrotics, cases of congestive cardiac failure, hepatic cirrhosis with failure and in eclampsia—examples of secondary aldosteronism. A transient form of this disorder occurs also in the first 24 hours after major surgical procedures. The primary form of the syndrome was described by Conn and Louis as recently as 1954. Most of the patients treated so far have been cured by removal of an adenoma of the adrenal cortex.

In the differential diagnosis of Addison's disease it is interesting to consider the problem posed by chronic arsenical poisoning, since this can in many ways closely simulate the picture of Addison's disease which, if complicated by an alcoholic peripheral neuritis, may be differentiated only by toxicological investigations.

The clinical section of the *Atlas* concludes with a chapter on miscellaneous conditions not clearly endocrine in nature, but which often need to be considered in the evaluation of an endocrinopathy. An *Appendix* contains useful tables on height, weight and growth curves. There is also a table of bone age taken from Engelbach and McMahon (1924). Since that date we have become more conscious of the marked sex difference in bone age. In the wrist and in the elbow, females may be as much as 2 years ahead of males in the appearance of certain ossification centres.

A more recent study of data dealing with bone age and taking into account the now well-established sex differences is an important modification which should be introduced into Engelbach and McMahon's Table in future editions of the *Atlas*.

Throughout this volume close attention is paid to the problem of treatment, whether surgical or endocrine. This feature enhances the practical value of one of the most outstanding contributions made in this field in recent years.

CRYPTOCOCCOSIS

Cryptococcosis. By M. L. Littman, M.D., Ph.D., and Lorenz E. Zimmerman, M.D. 1956. (Pp. 182 + Index. With Figs. and 4 colour plates. 72s. 3d.). New York and London: Grune & Stratton.

This is a most useful monograph. The disease with which it deals is widely distributed throughout the world (unlike the majority of systemic mycoses) and comprehensive studies which serve both pathologist and clinician are few indeed. The serious nature of the disease, its protracted course and the difficulties of treatment create a demand for detailed studies of the infecting agent. Certainly this work has a bias towards the laboratory aspect, but this is inevitable in such a subject. However, there are also detailed discussions on the biology, immunology, clinical features, treatment and prognosis. An *Appendix* dealing with cultural methods and histological techniques is included, together with an imposing bibliography of over 500 references. The former features are exceptional in their detail and quality.

The monograph is written in an easy, clear style, and it is profusely illustrated in black and white as well as with colour plates of superlative standard.

This work will be read by pathologists and medical mycologists with interest and benefit, and it constitutes a worthy contribution by the authors who are already distinguished in this field.

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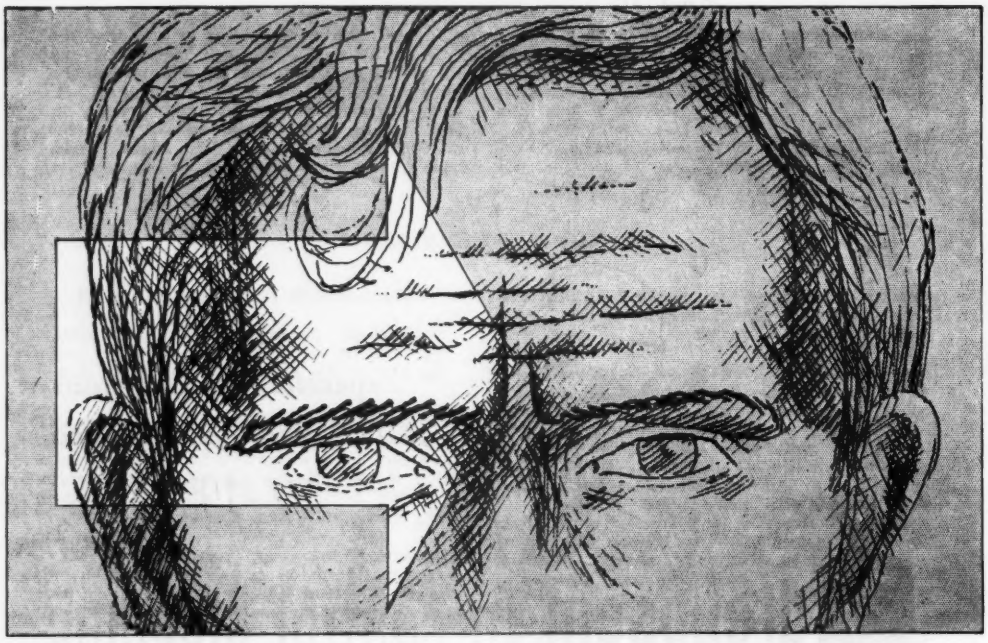

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1. Weiss, E., and English, O.S.: Psychosomatic Medicine, W. B. Saunders Co., Philadelphia, 1949 ,p. 358.

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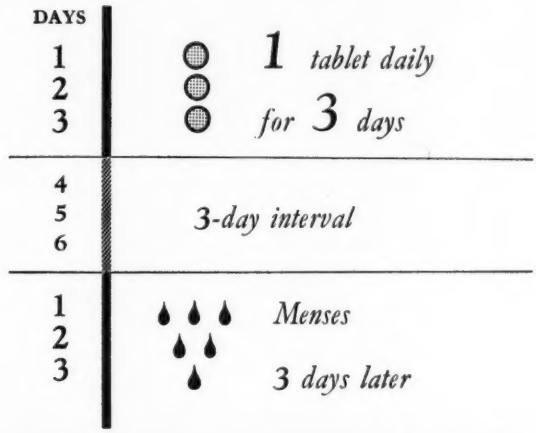

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